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IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Application of:)
A. LUCIW, ET AL.) Group Art Unit: 1813
No. 08/083,391) Examiner: M. Woodward
June 28, 1993) Attorney Docket No. 0035.008

HIV IMMUNOASSAYS USING GAG POLYPEPTIDES (AS AMENDED)

DECLARATION

able Commissioner of Patents
Trademarks
ngton, D.C. 20231

I, John A.T. Young, do hereby declare as follows:

1. I received my Ph.D. in Human Genetics from Imperial Cancer Research Fund
niversity College, London, United Kingdom in 1987 having previously received a B.S.
hemistry from the University of Dundee in 1983.
2. I am currently an Assistant Professor, Department of Microbiology and Molecular
ics, Harvard Medical School. My Curriculum Vitae is attached as Exhibit 1.
3. I have read and understand Luciw et al. application Serial No. 08/083,391 and
et al. application Serial No. 06/667,501 ('501) as well as the Office Action mailed
ary 20, 1996
4. The HIV nucleotide and amino acid sequences provided in the '501 parent
ation enabled one of ordinary skill in the art in October 1984 to identify HIV antigenic
es, i.e., peptides containing an immunogenic amino acid sequence. To demonstrate
performed a hydrophilicity analysis of the ARV-2 Env sequence, according to the Hopp
sol (Hopp 1981, Hopp 1983). The directions in Hopp, together with the hydrophilicity

ies given in Hopp 1981, permit a straightforward analysis that was easily within the skill of the art in October 1984. The confirmation of antigenicity was also within the skill of the art 384. An antigen could be screened by using it in an immunoassay such as a conventional or art immunoassay and testing it with sera of patients known to be infected. This screening process is the technique that is, in fact, disclosed in the Hopp references.

5. Employing the Hopp protocol, the most hydrophilic region of ARV-2 Env, was identified as residues 738-743 (ERDRDR). Peptides derived from HIV Env that contain these 10 acid residues are recognized by a proportion of AIDS patient antisera as demonstrated later actual tests. (Broliden 1992, Goudsmidt 1990, Kennedy 1986). The second-most hydrophilic region was identified as residues 653-658 (EKNEQE). Peptides containing this region of HIV Env are also recognized by sera from HIV infected individuals (Broliden 1992, Goudsmidt 1990, Krowka 1991). The third most hydrophilic region of ARV-2 Env, residues 733-738 (EEEGGE), overlaps the first hydrophilic region. Peptides containing this third region of Env are recognized by sera from HIV infected individuals. (Broliden 1992, Goudsmidt 1990, Kennedy 1986) The region containing residues 505-510 (QREKRA) was also identified as being highly hydrophilic. This finding was noted using the same computer analysis by Letti (1985). Peptides derived from HIV Env containing all or most of these residues are recognized by AIDS patient antisera (Broliden 1992, Kennedy 1987, Krowka 1991, Shcheryakova 1993, Palker 1987, Streckert 1992).

6. Employing the Hopp protocol, the most hydrophilic region of ARV-2 Gag, was identified as residues 102-107 (EKIEEE). Peptides derived from HIV Gag that contain these 6 amino acid residues are recognized by a proportion of AIDS patient antisera as demonstrated later actual tests. (Jiang 1992). The second-most hydrophilic region was identified as

s 109-114 (NKSKKK). Peptides containing this region of HIV Gag are immunogenic & recognized by sera from HIV infected individuals (Jiang 1992).

7. The HIV sequences provided in the '501 parent application also enabled one & any skill in the art in October, 1984 to identify antigenic HIV Gag linear epitopes by still & techniques. One other approach known in the art, was to generate one or a panel of synthetic peptides derived from the polypeptide sequence and test each peptide for & y reactivity. The generation of one or a panel of synthetic polypeptides from a single & was a routine matter in 1984.

8. A panel of eight peptides (each 13-15 amino acids in length) of interleukin-2 was & tested by Altman (Altman 1984) and a panel of five synthetic peptides (8 to 16 amino & & long) derived from adenovirus 19K and 53K proteins was generated by Green. (Green & In addition, Sutcliffe generated a panel of 12 peptides from MuLV polymerase gene & panel of 18 peptides from the rabies glycoprotein gene. (Sutcliffe 1983)

9. Prior to October 1984, those skilled in the art knew that a proportion of & bodies raised against native proteins could recognize epitopes contained on synthetic & is derived from a protein sequence (Rothbard 1984; Leach 1983) or contained on & ytic protein fragments (Lando 1982).

10. The open reading frame labeled "GAG", indicated by brackets in Fig. 4 of the & application precisely delineates the actual boundaries of the HIV Gag gene. & more, the N-terminal ends of two proteolytic fragments of the Gag precursor & protein (p25 and a p18) are correctly identified in Tables 1 and 2, and Figure 4 of the '501 & application. Based upon this information, one skilled in the art would have undoubtedly

ectly identified the exact parameters of the Gag precursor polyprotein from the '501
lication.

11. Based on the information described herein, those skilled in the art could have,
out undue experimentation, used the sequence of ARV-2 Gag provided in the '501
ication to generate peptides representing most of this HIV protein. These peptides could
have been tested using standard assays known in the art, and immunogenic regions of
Gag identified.

12. Cloning and expression in bacteria was a routine matter in October 1984
onstrated, *inter alia*, by Maniatis, of record, a well known and widely used laboratory
ual. Representatives of a number of publications reporting the recombinant production
roteins in prokaryotes are Stanley, *EMBO J.*, 3:1429-1434 (1984) and Ghrayeb, *EMBO*
:2437-2442 (1984).

13. The issue of intervening sequences is not relevant for retroviral Gag genes.
proteins are the products of "unspliced" retroviral mRNA transcripts, i.e., intervening
uences are not removed from transcripts that encode any known retroviral Gag
proteins.

14. The fact that Maniatis chose not to include eukaryotic expression systems in his
2 laboratory manual bears absolutely no relevance on the question of whether eukaryotic
ession systems were being widely used by October of 1984. Indeed a number of labs had
cessfully expressed a variety of recombinant genes in eukaryotic cells at that time
uding α -globin by workers including Maniatis (Mellon, *Cell*, 27:279-288 (1981); Wigler,
, 16:777-785 (1979). Other eukaryotic expression systems include human proinsulin
iao-Ping, *Cell*, 35:531-538 (1983), corticotropin- β -lipotropin (Mishina, *EMBO J.*, 1:1533-

1982), β -globin and δ -globin (Humphries, *Cell*, 30:173-183 (1982). Once again, still can be documented if needed.

15. A number of studies have confirmed that HIV encoded factors are not required expression of the HIV Gag gene in a variety of different systems. These include al expression systems, yeast expression systems and baculovirus-based insect cell sion systems (e.g. Wagner, *Archives of Virol.*, 127:117-137 (1992); Luban, *J. Virol.*, 2-3212 (1991); Komiyama, *AIDS*, 5:411-419 (1989); Chazal, *J. Virol.*, 68:111-122 Geysen, *Cell*, 59:103-112 (1989); Luban, *J. Virol.*, 66:5157-5160 (1992); Jacobs, 79:71-81 (1992).

16. One of ordinary skill in the art in 1984 understood the term "synthetic peptide" a peptide prepared by chemical synthesis. The term "synthetic" was used to describe synthesized by chemical means in numerous publications prior to the October 31, filing date of parent application Serial No. 06/667,501. Representative publications (re still others) include Altman 1984, Barkas 1984, Bellet 1984, Dale 1983, Green 1983, 982, Hirayama 1982, Jacob 1983, Jolivet 1983, Lieu 1975, Morrow 1983, Morrow 1984, 1983, Pacella 1983, Rothbard 1984, Rougon 1984, Sherwood 1983, Shi 1984, Sutcliffe Tamura 1982, and Wabuke-Bunoti 1984.¹ The articles were published in a variety of own journals, including those read by a general scientific audience (e.g., *PNAS* and e) as well as those read mainly by virologists and immunologists (e.g., *Journal of y and Molecular Immunology*). These are the journals that one skilled in the art would ected to review.

¹The full citation for each of the references cited in this declaration is included in : 2.

17. Following 1984, the term "synthetic" was still understood by those skilled in the art to mean a peptide synthesized by chemical means. This is illustrated by the following evidence taken from Chapter 5 under the sub-heading "Synthetic peptides" of a widely-quoted laboratory research manual (Harlow, E., and D. Lane. 1988, Antibodies: a laboratory manual. Cold Spring Harbor Laboratory, Cold Spring Harbor, N.Y.): "Peptides are normally synthesized using the solid-phase techniques pioneered by Merrifield (1963)." The term is so-understood today.

18. Prior to October 31, 1984 one skilled in the art was fully capable of synthesizing peptides of considerable length. Specific examples of synthetic polypeptides containing as many as 40 amino acids were reported in the art prior to October 31, 1984. Ten of the above-mentioned articles (Altman 1984, Barkas 1984, Dale 1983, Hirayama 1982, Jacob 1983, Jar 1983, Rothbard 1984, Shi 1984, and Wabuke-Bunoti 1984) report synthetic peptides (peptides made by chemical synthesis) having lengths of from 15 to 24 amino acids and one article (Bellet 1984) reports a 37 amino acid synthetic peptide. Reid (1981) employed a 31 amino acid synthetic peptide, while Puett (1982) employed a 40 amino acid synthetic peptide.

19. A number of alternate assays were known to those skilled in the art in October 1984 by which to identify antigenic polypeptides. These included immunoblotting (e.g., Abbott, *J. Virol.*, 49:379-385 (1984); Mertens, *Virol.*, 129:431-442 (1983); Fujinami, *PNAS*, 78:2346-2350 (1983); Kandor-Koch, *J. Cell Biol.*, 97:644-651 (1983), and immunoprecipitation (e.g., Crawford, *J. Virol.*, 29:587-596 (1979); Silva, *J. Virol.*, 35:766-774 (1980); Abrams, *Cell*, 31:427-439 (1982); Rassoulzadegan, *PNAS*, 80:4354-4358 (1983). Still others can be implemented if needed.

20. I have reviewed in detail Montagnier, *Science*, 225, 63-66 (July, 1984) and Bach et al., *Science*, 224, 503-505 (May, 1984). In my opinion these articles would not have enabled one skilled in the art to prepare a synthetic or recombinant HIV Gag polypeptide probe for use in an immunoassay without undue experimentation. I conclude this for the following reasons:

- a) These articles did not provide any HIV nucleotide or amino acid sequence information.
- b) Although HIV proteins were purportedly identified by immunoblotting in these publications, a person of ordinary skill in the art would not have been able to produce sufficient quantities of any of these viral proteins for sequencing. Sufficient quantities could not have been produced because cultures of primary human cells failed to produce significant quantities of HIV, as the virus is cytopathic and rapidly killed the infected virus-producing cells. Therefore, a person of ordinary skill in the art, attempting to generate sufficient quantities of HIV proteins for detailed characterization, would have i) had to obtain an appropriate established cell line known to produce HIV, and ii) had to have a knowledge of the precise conditions required for infecting these cells and for maintaining the infected cells for long periods of time in culture.
- c) By October 31, 1984, the Gallo and Montagnier groups had reported cell lines that could be used to produce significant levels of HIV (Popovic 1984, Montagnier 1984). Gallo and Montagnier were world leaders in HIV research at the time, and thus can hardly be considered to be of "ordinary skill in the art". At the time of the '501 application date, the precise origin of the cell line used by the Gallo group had not been disclosed (Popovic 1984). The Montagnier group used cells generated by fusion

between HIV producing primary T cells and EBV-transformed B-cells (Montagnier 1984). It would not have been possible for a scientist of ordinary skill in the art to have used the same technique to produce cell lines that were identical to those described by the Montagnier group. Even if a scientist of ordinary skill in the art had attempted to obtain the cells described by the Gallo and Montagnier groups, I am not aware of any evidence that these cell lines were being distributed freely to the public at the time of the '501 application date. Furthermore, the precise culture conditions required for maintaining HIV-infected cells in culture had not been disclosed.

21. I have also reviewed in detail Chang U.S. application Serial No. 659,339 filed October 10, 1984 including the partial DNA sequence of Figure 3. Although Chang represents the Figure 3 sequence "encompasses the env region" (p. 5, lines 1-2), that is incorrect. In fact, the Figure 3 sequence contains a portion of the *pol* gene, the *sor* gene and only approximately one-third of the envelope gene. The Chang Figure 3 does not contain any of the Gag sequence.

I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code and that such willful false statements may jeopardize the validity of the application or any patent issuing thereon.

Date: 4/22/97

By: John A.T. Young
John A.T. Young

CURRICULUM VITAE

~~RESUME~~

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Marital Status: Married to Dr. Caroline Alexander
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ACADEMIC APPOINTMENTS

2 to 1995 Assistant Professor
Department of Microbiology and Immunology
University of California, San Francisco
San Francisco, California

2 to 1995 Assistant Investigator
Gladstone Institute of Virology and Immunology
San Francisco General Hospital
San Francisco, California

2 to 1995 Member, Program in Biological Sciences (PIBS)
Cell Biology Program
University of California, San Francisco

2 to 1995 Member, Biomedical Sciences Program
University of California, San Francisco

15 to Assistant Professor
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Harvard Medical School

15 to Member, Biological and Biomedical Sciences Program,
Harvard Medical School

15 to Member, Committee on Virology, Harvard Medical School

1995 to Member, Board of Tutors in Biochemical Sciences, Harvard University

POSTDOCTORAL TRAINING

1987-1989 EMBO Postdoctoral Fellow
Department of Microbiology and Immunology
University of California, San Francisco
Advisor: Harold B. Varmus, M.D.

1989-1992 Arthritis Foundation Postdoctoral Fellow
Department of Microbiology and Immunology
University of California, San Francisco
Advisor: Harold B. Varmus, M.D.

EDUCATION

1983 University of Dundee
Dundee, United Kingdom
B.Sc., Biochemistry (First Class Honours)

1987 Imperial Cancer Research Fund and University College
London, United Kingdom
Ph.D., Human Genetics
Thesis: Expression and Polymorphism of H.I.A-D Region Genes
Ph.D. Advisor: John Trowsdale, Ph.D.

TEACHING EXPERIENCE

1992 Co-organizer
Introduction to Cell Biology course
Medicine 412, UCSF

Discussion Leader
Cell Biology Course 212, UCSF

1993 Lecturer
The Biology of AIDS
Biomedical Sciences Minisymposium, UCSF

1993 Discussion Leader
Tissue Organization and Morphogenesis course
Biomedical Sciences 210, UCSF

Discussion Leader
Molecular Biology of Animal Viruses course
Microbiology 208, UCSF

1994 Lecturer, The Biology of Virus Infection course
Microbiology 208, UCSF

96 Lecturer, Microbiology 201, Harvard Medical School
(4 lectures, 9 discussion groups)

97 Co-director, Virology 200, Harvard Medical School

97 Lecturer, Virology 200, Harvard Medical School (3 lectures)

COMMITTEES

92 to 1995 Member, Dean's Advisory Committee to the UCSF AIDS Clinical Research Center

93 to 1995 Member, Executive Committee of the UCSF Biomedical Sciences Program

93 to 1995 Member, UCSF Student Research Committee

96 to Member, Virology Admissions Committee, Harvard Medical School

96 to Member, Division of Medical Sciences Curriculum Committee, Harvard Medical School

RAINEES

1992 to 1995 Kurt Zingler
Ph.D. Thesis Student
Immunology Program, UCSF

Jürgen Brojalsch, Ph.D.
Postdoctoral Fellow

Carole Bélanger, Ph.D.
Postdoctoral Fellow
Fonds de la Recherche en Santé du Québec

1993 to 1995 Lynn Connolly
M.D., Ph.D. Thesis Student
Medical Scientist Training Program, UCSF

Morgan Seakins, M.D.
Clinical Research Fellow
Universitywide AIDS Research Program

1996 to Heather B. Atkins
Ph.D. Thesis Student
Curriculum on Virology, Harvard Medical School

1996 to Vincent Solomon
Ph.D. Thesis Student
Biological and Biomedical Sciences, Harvard Medical School

PUBLICATIONS

1. Trowsdale, J., Young, J.A.T., Kelly, A.P., Austin, P.J., Carson, S., Mcunie, H., So, A., Ehrlich, H.A., Spielberg, R.S., Bodmer, J., and Bodmer, W. (1985) Structure, sequence and polymorphism in the HLA-D region. *Immunol. Rev.* 85:135-173.
2. Young, J.A.T. and Trowsdale, J. (1985) A processed pseudogene in an intron of the HLA-DP β 1 chain gene is a member of the ribosomal protein L32 gene family. *Nucl. Acids Res.* 13:8883-8891.
3. Trowsdale, J., Austin, P., Carson, S., Kelly, A., Lamb, J., and Young, J.A.T. (1985) Cloned HLA-D genes: Characterisation and approaches to expression and analysis of function. In: *Human T-cell Clones* (M. Feldmann, J.R. Lamb, and J.N. Woody, eds.), The Human Press, pp 49-57.
4. Bodmer, W.F., Trowsdale, J., Young, J., and Bodmer, J. (1986) Gene clusters and the evolution of the major histocompatibility complex. *Phil. Trans. R. Soc. Lond.* 312:303-315.
5. Young, J.A.T., Wilkinson, D., Bodmer, W.F., and Trowsdale, J. (1987) Sequence and evolution of HLA-DR7- and HLA-DRw53-associated β chains. *Proc. Natl. Acad. Sci. USA* 84:4924-4933.
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9. Young, J.A.T. and Trowsdale, J. (1990) The HLA-DNA gene is expressed as a 1.1 kb mature mRNA species. *Immunogenetics* 31:386-388.
10. Young, J.A.T., Bales, P., Willert, K., and Varmus, H.E. (1990) Efficient incorporation of human CD4 protein into Avian Leukosis Virus particles. *Science* 250:1421-1423.
11. Young, J.A.T., Bales, P., and Varmus, H.E. (1993) Isolation of a chicken gene that confers susceptibility to infection by subgroup A avian leukosis and sarcoma viruses. *J. Virol.* 67:1811-1816.
12. Bales, P., Young, J.A.T., and Varmus, H.E. (1993) A receptor for subgroup A Rous sarcoma virus is related to the low density lipoprotein receptor. *Cell* 74:1043-1051.
13. Connolly, L., Zingler, K., and Young, J.A.T. (1994) A soluble form of a receptor for subgroup A avian leukosis and sarcoma viruses (ALSV-A) blocks infection and binds directly to ALSV-A. *J. Virol.* 68:2760-2764.
14. Young, J.A.T., Bales, P.R., and Varmus, H.E. (1994) A protein related to the LDL receptor is a cellular receptor specific for subgroup A-avian leukosis and sarcoma viruses. In:

Receptor-mediated Virus Entry into Cells. (E. Wimmer, ed.) Cold Spring Harbor Laboratory, Cold Spring Harbor, NY.

15. Young, J.A.T. (1994) The replication cycle of HIV-1. In: *The AIDS Knowledge Base.* (Cohen, P., Sande, M., Volberding, P., et al., eds.) Little Brown, New York, NY.
16. Federspiel, M.J., Rules, P., Young, J.A.T., Varmus, H.E., and Hughes, S.H. (1994) A system for tissue-specific gene targeting: Transgenic mice susceptible to subgroup A avian leukosis virus-based retroviral vectors. *Proc. Natl. Acad. Sci. USA* 91: 11241-11245
17. Bélanger, C., Zingler, K., and Young, J.A.T. (1995) Importance of cysteines in the LDLR-related domain of the ALSV-A receptor for viral entry. *J. Virol.* 69: 1019-1024.
18. Zingler, K., Bélanger, C., Peters, R., Agard, D. and Young, J.A.T. (1995) Identification and characterization of the viral interaction determinant of the ALV-A receptor. *J. Virol.* 69: 4261-4266
19. Zingler, K. and Young, J.A.T. (1996) Residue Trp-48 of Tva is critical for viral entry but not for high-affinity binding to the SU glycoprotein of Subgroup A avian leukosis and sarcoma viruses. *J. Virol.* 70: 7510-7516
20. Young, J.A.T. (1996) The replication cycle of HIV-1. In: *The AIDS Knowledge Base.* (Cohen, P., Sande, M., Volberding, P., et al., eds.) Little Brown, New York, NY in press.
21. Brojalsch, J., Naughton, J., Rolls, M.R., Zingler, K. and Young, J.A.T. (1996) CAR1, a TNFR-related protein is a cellular receptor for cytopathic avian leukosis and sarcoma viruses and mediates apoptosis. *Cell* 87: 845-855.

INVITED PRESENTATIONS (Meetings)

Invited Chair, Roundtable Discussion on Gene Therapies for AIDS. Second Annual NIH National AIDS Cooperative Drug Discovery and Development Meeting. California. 1988.

Invited Speaker, Banbury Conference on Receptor Mediated Virus Entry into Cells, Cold Spring Harbor Laboratory, 1991.

Invited Speaker, Keystone Symposium on Molecular Biology of Human Pathogenic Viruses. California, 1993.

Invited Speaker, Fifth Workshop on Pathogenesis by Non-acute Retroviruses. France, 1993.

Invited Speaker, Workshop on Immunology and Developmental Biology of the Chicken. Basel Institute of Immunology, Switzerland, 1994.

Invited Speaker, Sixth Workshop on Pathogenesis of Animal Retroviruses. Philadelphia 1994

Invited Speaker, Seventh Workshop on Pathogenesis of Animal Retroviruses. Seattle 1995.

Chair, Session on Receptors, Entry and Uncoating, Retroviruses Meeting at Cold Spring Harbor Laboratory, New York, 1996

Invited Speaker, FASEB Summer Conference on Principles in Viral, Bacterial, Fungal and Protozoan Pathogenesis. Colorado, 1996

Invited Speaker, FASEB Summer Conference on Virus Assembly, Vermont, 1996

Invited Symposium Speaker, American Society for Virology Symposium. Montana 1997

Invited Speaker, Cleveland Virology Symposium, 1997

Invited Speaker, Animal Cells and Viruses Gordon Conference, 1997

INVITED PRESENTATIONS (Institutions)

Department of Microbiology and Immunology, Penn State Medical Center, Hershey Pennsylvania, October 1996.

Department of Molecular Microbiology, Washington University at St. Louis School of Medicine, St. Louis, November 1996

Department of Microbiology, New York University Medical School, New York, January 1997

OTHER PRESENTATIONS

An attempt to specifically alter retroviral tropism using EGF-envelope chimeras. J.A.T. Young, P. Bales, H. Varma. Poster presentation at Cold Spring Harbor RNA Tumor Viruses meeting. May 1988.

Transfer of susceptibility to ALSV infection into mammalian cells with chicken DNA. P. Bales, J.A.T. Young, and H.B. Varma. Talk at Cold Spring Harbor RNA Tumor Viruses meeting. May 1989.

The human CD4 protein is efficiently incorporated into ALV particles. J.A.T. Young, P. Bales.

K. Willert, and H.E. Varmus. Talk at Cold Spring Harbor RNA Tumor Viruses meeting. May 1990.

A DL receptor-related protein is the subgroup A ALV receptor. P. Bates, J.A.T. Young, H.E. Varmus. Talk at Cold Spring Harbor RNA Tumor Viruses meeting. May 1992.

Functional characterization of the subgroup A-Avian Leukosis Virus (ALV) receptor gene: Low levels of receptor expression are limiting for virus infection. J.A.T. Young, P. Bates, H.E. Varmus. Talk at Cold Spring Harbor RNA Tumor Viruses meeting. May 1992.

Mutational analysis of the cellular receptor for subgroup A-ALSV. K. Zingler, C. Bclanger, J.A.T. Young. Talk at Cold Spring Harbor Retroviruses meeting. May 1994.

A soluble version of the subgroup A-ALSV receptor blocks infection and binds directly to ALSV-A. L. Connolly, K. Zingler, J.A.T. Young. Talk at Cold Spring Harbor Retroviruses meeting. May 1994

An assay system to determine the relative levels of intermediate and complete DNA forms of HIV-1 DNA following infection. M. Jenkins, J. Naughton, J.A.T. Young. Poster presentation at Cold Spring Harbor Retroviruses meeting. May 1994

A putative receptor for cytopathic subgroups of ALSVs is a member of the Fas/TNFR protein superfamily. J. Brofatsch, J. Naughton, J.A.T. Young. Talk at Cold Spring Harbor Retroviruses meeting. May 1996.

Evidence that residue Trp-48 of TVA is involved at a step of viral entry other than binding the SU glycoprotein of subgroup A avian leukosis and sarcoma viruses. K. Zingler, J.A.T. Young. Talk at Cold Spring Harbor Retroviruses meeting. May 1996.

G N23

Characterization of ALSV-A Env/Receptor Interactions

NIH: IR29CAAI62000-01A1

\$615,301, July 1994 to June 1999

An Attempt to Target Retrovirus Vectors to Cells Expressing HIV-1 Envelope Proteins
AIDS Clinical Research Center, UCSF
One-year grant (\$25,000). Funded January 26, 1994

Milan Fund (\$12,000) Harvard Medical School, July 1995

Characterizing the Mechanisms of ALSV Entry into Cells

NIH: IRO1CA70810-01

\$ 985,949, July 1996 to June 2000.

OUTSIDE ACTIVITIES

1995 to present Consultant, Chiron Corporation, Emeryville, California

1995 to present Consultant, Vaccines and Related Biological Products Advisory Committee, Food and Drug Administration.

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Re Application of:)
JL A. LUCIW, ET AL.) Group Art Unit: 1813
al No. 08/083,391) Examiner: M. Woodward
d: June 28, 1993) Attorney Docket No. 0035.009

**HIV IMMUNOASSAYS USING SYNTHETIC
ENVELOPE POLYPEPTIDES (AS AMENDED)**

JOHN A.T. YOUNG BIBLIOGRAPHY

Abrams, et al.	Cell, 29:427-439 (1982)*
Altman, et al.	PNAS, 81(7):2176-2180 (1984)
Barkas, et al.	Eur. J. Biochem., 143(2):309-314 (1984)
Ballet, et al.	Endocrin., 115(1):330-336 (1984)
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Hirayama, et al.	Immuno., 46(1):145-154 (1982)
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Hopp, et al.	Mol. Immuno., 20(4):483-489 (1983)
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Jiang, et al.	J. Acquired Imm. Def. Synd., 5:382-390 (1992)
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Kandor-Koch, et al.	J. Cell Biol., 97:644-651 (1983)*
Kennedy, et al.	Science, 231:1556-1559 (1986)
Kennedy, et al.	J. Biol. Chem., 262(12):5769-5774 (1987)
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Krowka, et al.	Clin. Immunol. & Immunopath., 59(1):53-64 (1991)
Lando, et al.	J. Immunol., 129(1):212-216 (1982)
Leach, et al.	Biopolymers, 22:425-440 (1983)
Lieu, et al.	Immuno., 29(6):1133-1143 (1975)
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Copies of the references marked (*) will be supplied with the executed copy of
the declaration. The remaining references were submitted with the Young
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